A MECHANICAL PUMP-OXYGENATOR FOR DIRECT VISION REPAIR OF ATRIAL SEPTAL DEFECTS

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THE desire for a direct vision surgical approach to intracardiac defects has stimulated the development of mechanical pump-oxygenators. The various extracorporeal circuits now available have been designed to deliver pulsatile or continuous flows of blood oxygenated by filming,[1, 2] bubbling,[3] dialysis,[4] and passage through the isolated lung.[5] Failure of many of these by-pass systems to gain wider clinical application has been due to the potential hazards associated with the use of them. Air embolism of extra- and intracardiac origin and hemorrhage constitute the principal dangers to the recipient.

The extracorporeal circuit described in this report, a modification of an earlier model,[6] has been used extensively in experimental procedures to investigate these problems.[7, 8] Additional studies were conducted to re-define the acute metabolic changes in animals undergoing total by-pass, since the physiologic alterations suggested by previous results were not in accord with our current concepts. The material in this presentation deals with our experience in the creation and repair of interatrial septal defects in 31 dogs and the successful application of the pump-oxygenator to a clinical case.

METHOD

Mongrel dogs, weighing 8 to 35 Kg., were anesthetized with intravenous pentobarbital sodium, intubated, and placed on a mechanical respirator. Surgery was performed aseptically. The chest was entered through the bed of the right fifth rib (first 12 dogs) or transsternally in the fourth intercostal space (19 dogs). The azygos vein was ligated in continuity. The left subclavian artery was exposed and divided distally. After the pericardium had been widely incised, the inferior and superior venae cavae were isolated extrapericardially and surrounded by sling ligatures. Hemostasis was obtained by the use of electrocautery. Heparin (Connaught Laboratories), 2.5 mg. per kilogram, was administered intravenously at the completion of all dissections.

During this initial preparation, the extracorporeal circuit, which has been reported previously in detail,[7] was assembled (Fig. 1). The flowmeter was

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an electromagnetically recording rotameter.[10] The rate of venous outflow was recorded on a Sanborn Poly-Viso. The venous blood was delivered to rubber jets placed near the axis of the oxygenator and filmed on four rotating discs. Venous drainage was assisted by a siphon effect equal to the vertical distance between the venae cavae and the openings of the jets (*A* to *B* in Fig. 1); this measured 40 cm. in most animals. The oxygenator tank was continuously flushed by 100 per cent oxygen. The bubble trap contained stainless

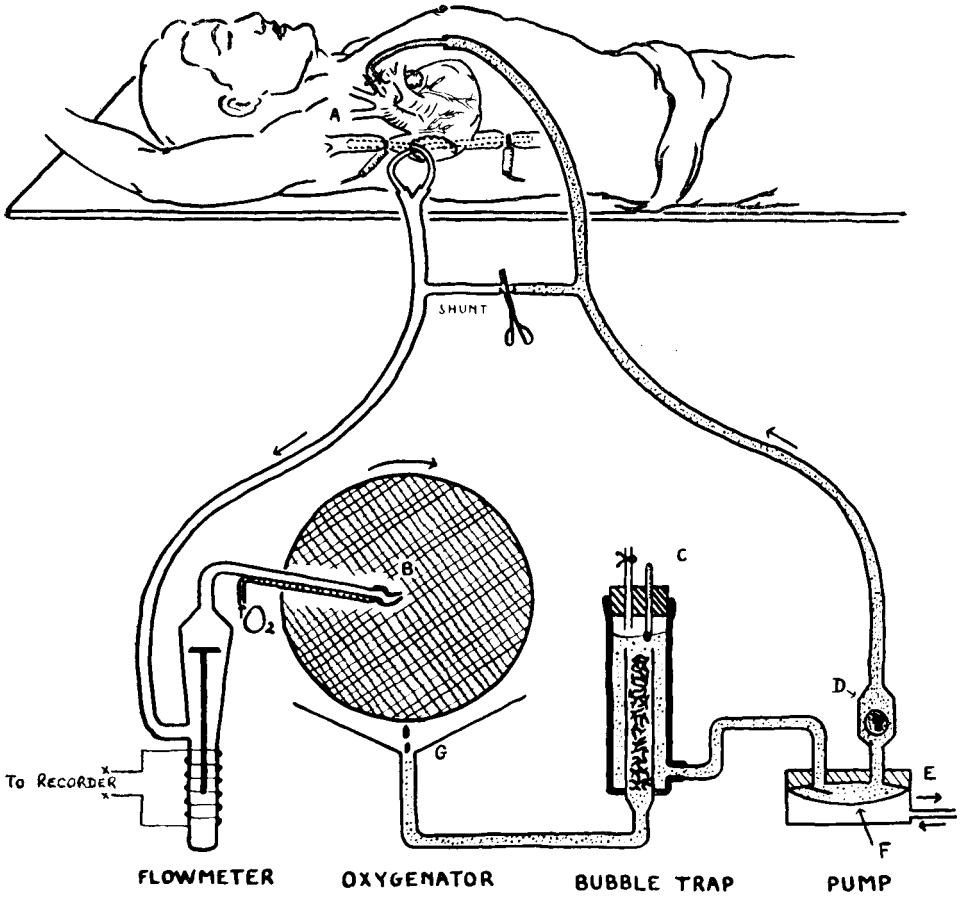

Fig. 1.—Diagram of pump-oxygenator assembly for total body perfusion. *A*, Catheters in place in the left subclavian artery and in the superior and inferior venae cavae. *B*, Rubber jets film the blood on rotating discs above oxygen inlet. C, Outlet for removing air accumulated in bubble trap and thermometer for recording blood temperature. *D*, Nylon shuttle valve prevents regurgitation into pump. *E*, Compressed air from solenoids motivates diaphragm, *F*, of pump which is filled by hydrostatic pressure of blood column, *G*.

steel scouring sponge, coated with Antifoam-A (Dow Corning), within the inner cylinder.[3] It served effectively both as a bubble remover and as a filter for fibrin. In no instance in these experiments was the extracorporeal origin of air embolism implicated. The modified Dale-Schuster pumps[11] were activated by compressed air regulated by solenoid valves. A pulsatile stream, 14 to 17 ml. per stroke, was delivered to the systemic circulation. Filling of

the pumps was dependent upon the displacement of rubber diaphragms by the hydrostatic pressure of the column of blood (*G* in Fig. 1) from the oxygenator. "Gravity filling" eliminated the negative phase in the pumping cycle and prevented air above the blood level in the oxygenator from entering the arterial side of the extracorporeal circuit. The pumps were therefore self-regulatory, accelerating when the influx of blood was increased and slowing as the flow decreased. This minimized gross fluctuations in the holdup volume.[12] For any single perfusion, the stroke output was fixed and the minute volume varied directly as the rate. In anticipation of high flows in large subjects (over 40 Kg.), the capacity of the system was increased by incorporating two bubble traps and using two larger pumps with stroke outputs of 85 ml. The principal limiting factor in achieving these high flows was the caliber of the cannula capable of being threaded into the artery of the recipient.

Freshly drawn, unmatched blood, heparinized with 30 mg. per 1,000 ml., was obtained from donor dogs for priming the extracorporeal circuit (1,100 ml.) and replacement of operative losses. The femoral vein and artery were used for drainage and return of blood in the first 9 animals. In subsequent procedures, the left subclavian artery was cannulated with a plastic catheter (OD – 14 Fr.).* Similar catheters were placed in the superior and inferior venae cavae through the right auricular appendage with the tips positioned distal to the sling ligatures. A shunt (see Fig. 1) connecting the efferent and afferent limbs of the extracorporeal circuit provided a method for removing residual air bubbles from the system before perfusion.

Blood pressure in the femoral artery and venous pressure in the external jugular vein were monitored by Statham strain gauges recording on a Sanborn Poly-Viso. The electrocardiogram was observed on an oscilloscope throughout the procedure. Prior to and again at the end of the by-pass, blood samples were obtained for determinations of the pH, CO_2, oxygen content of arterial and venous blood, hemoglobin, hematocrit, free plasma hemoglobin,[13] total protein, albumin, fibrinogen, pyruvic acid, plasma chloride, and protamine titre.[14] The analytical methods used for blood chemistry have been recorded elsewhere.[9]

The animal was placed on partial perfusion by occluding the shunt and opening the cannulae (see Fig. 1). Total perfusion was obtained by tightening the sling ligatures about the venae cavae. The heart was allowed to empty for 15 seconds. A 5 centimeter incision was made in the wall of the right atrium from the junction with the inferior vena cava parallel to the atrioventricular groove (Fig. 2). The coronary sinus, tricuspid valve, and crista terminalis were identified. A 2 to 2.5 centimeter incision was made in the interatrial septum. This was confirmed by the appearance of arterialized blood. Rapid suction was used to remove blood draining into the operative site from the left atrium, coronary sinus, and anterior cardiac veins. Frequently, this was returned to the extracorporeal circuit as losses accumulated. The septal incision was closed with interrupted silk sutures and the right atrial wall with a continuous silk suture.

*A beveled curved metal cannula was used for arterial cannulation in the first 17 animals.

Throughout the by-pass, efforts were made to maintain the blood volume at pre-perfusion levels. As they occurred, suction and other operative losses were corrected via the arterial side of the circuit by direct replacement into the oxygenator. In the presence of hypervolemia, a measured quantity of blood was permitted to drain back into the oxygenator. Size of the heart and venous and arterial pressures were guides to the restoration of a normal circulating volume. Protamine (diluted twentyfold with five per cent glucose in distilled water) was administered intravenously over a 15-minute period following decannulation. The dose was dictated by the protamine titer obtained at the end of perfusion. Blood losses incurred subsequent to this stage in the procedure were replaced by direct transfusions of fresh whole blood.

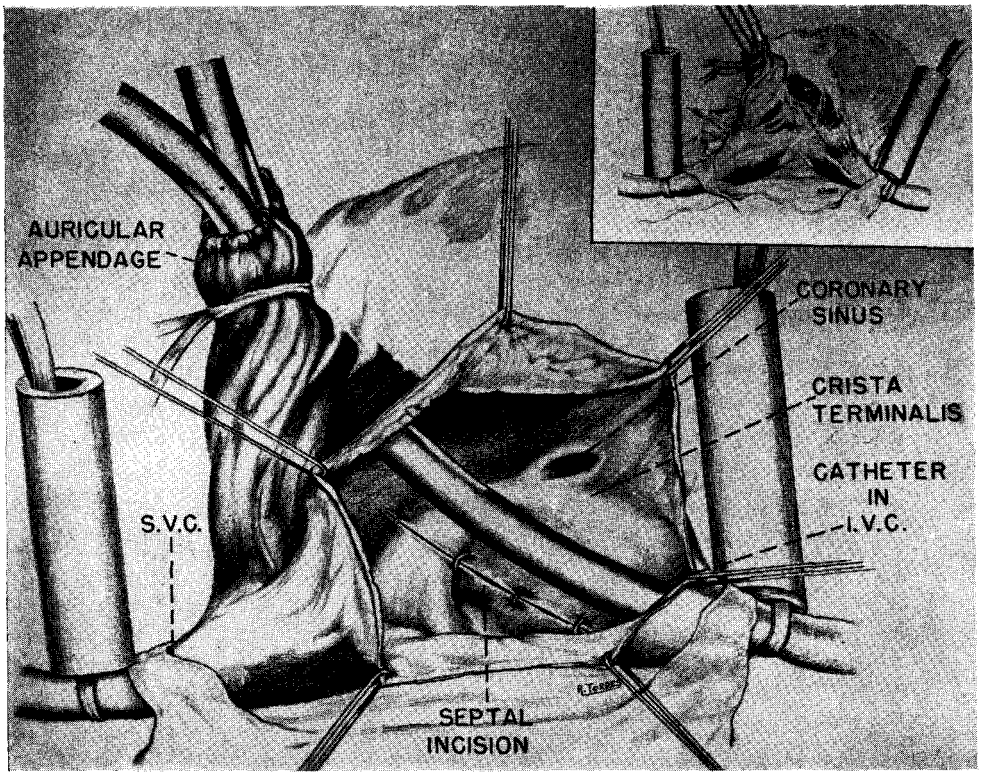

Fig. 2.—Exposure of the interatrial septum through the right atrium. The catheter can be displaced to facilitate the approach to the septum. *Inset;* Type of incision employed in atriotomy.

Large-bore thoracostomy tubes were placed in both pleural cavities and connected to waterseals. The pericardium was loosely approximated and the chest wound closed in layers. Postoperatively, the animals were observed with regard to adequacy of ventilation, chest drainage, body weight, blood pressure, rectal temperature, and reaction from anesthesia. To correct the mild hypothermia produced during the by-pass, the animals were rewarmed on blankets.* Vigorous efforts were made to evacuate blood and air from the pleural

*Therm-O-Rite Products Corp., Buffalo, N. Y.

cavity and encourage lung expansion. Bleeding from the chest was promptly corrected with whole blood transfusions. At regular intervals, fractional protamine titrations were obtained and appropriate doses of protamine administered until the clotting time returned to normal. Persistent chest drainage in the presence of a normal clotting time and absence of circulating heparin was an indication for re-exploration in one animal in which bleeding from the right atrial appendage was corrected. A normal circulating volume at the end of a procedure was reflected in a favorable comparison of pre- and postoperative body weights.

Three techniques were employed to prevent embolization of air through the incised interatrial septum. In 13 dogs a multiperforated vent was placed into the left ventricle through a stab wound in the avascular portion of the apex as recommended by Miller and his co-workers.[15] Aspirated blood and air under a negative pressure of –10 to –20 cm. of water, were returned directly to the oxygenator. In 12 dogs, an attempt was made by positioning the animals to preserve a continuous "lake" of blood or saline solution over the incised septum. Five degrees of Trendelenburg position were used to prevent air emboli from reaching the brain.[16] In 6 animals, ventricular fibrillation was induced with a shock of a 6-volt 60-cycle alternating current through two closely spaced electrodes applied to the ventricular epicardium.[17] Defibrillation was accomplished while the animal was on total perfusion with a countershock of 125 volts for about 0.1 second.

RESULTS

Thirty-one dogs were perfused for periods of 17 to 63 minutes in which time an interatrial septal defect was created and repaired. Rectal temperatures fell progressively during the by-pass. The average decrease was 5.5° C. and varied inversely with the size of the animal and directly with the length of perfusion. In experimental procedures no attempt was made to prevent this decline.

Perfusion pressures (femoral artery pressure) varied between 80 and 100 mm. Hg systolic and 30 and 50 mm. Hg diastolic. Insufficiencies in the circulating volume resulted in a fall in perfusion pressure. Pressure tracings during the perfusion showed secondary waves following ventricular systole. They were most marked in the diastolic phase of the pumping cycle and were abolished by ventricular fibrillation.

The results of analysis of blood oxygen and flows are summarized in Table I. Flows were measured by two methods. The pump rate multiplied by the

TABLE I. OXYGENATION OF BLOOD AND FLOWS AT END OF PERFUSION

	ARTERIAL		VENOUS				
	O_2 CONTENT (C.C./100 ML.)	O_2 SATURATION (PER CENT)	O_2 CONTENT (C.C./100 ML.)	O_2 SATURATION (PER CENT)	A-V DIFF. (C.C./100 ML.)	FLOW (ML./KG./MIN.)	O_2 CONSUMED (C.C./KG./MIN.)
Average	19.63	100.41	8.26	41.95	11.38	33	3.4
S.D.	±2.76	±4.75	±4.02	±14.35	±3.33		
Range							
minimum	12.52	93.05	2.65	17.34	4.23	15	1.2
maximum	23.83	108.74	15.34	62.90	16.71	55	6.2

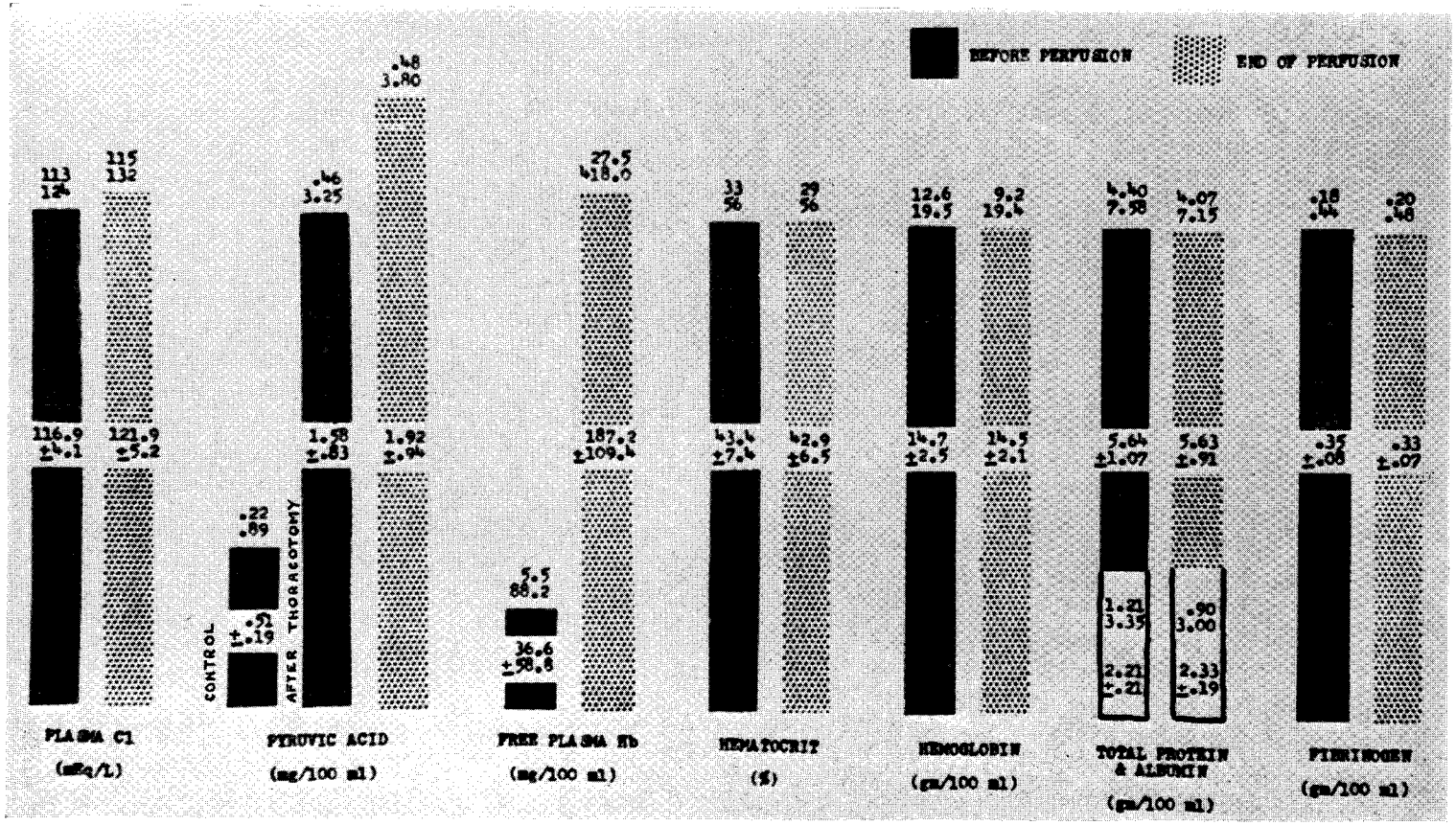

Fig. 3.—Results of blood determinations. (Figures at top of column represent the range of the average value which appears in the middle of the column with the standard deviation.)

stroke output corresponded with the flows recorded by the flowmeter. For 31 dogs this averaged 33 ml. per kilogram per minute (20 per cent of basal cardiac output[18]) and would place this pump-oxygenator in the low flow category. With this reduced turnover of blood, an average of 3.4 c.c. of oxygen per kilogram per minute was delivered to the tissues. This was 40 per cent of the assumed basal oxygen consumption and was attributed to the increase in arteriovenous difference (coefficient of oxygen utilization[19]). The highest oxygen uptake in this series was 102 c.c. per minute in a 34-kilogram animal with a flow of 940 c.c. per minute. This was well within the maximum oxygenating capacity of 130 c.c. per minute established for the rotating disc (four screens) oxygenator at a flow of 2 L. under the standard conditions set forth by Gimbel and Engelburg[12]; raising the oxygen saturation of hemoglobin from 60 per cent to 90 per cent.

In the first ten perfusions, a single phase, positive pressure, mechanical respirator[20] was used. The remaining experiments were conducted with an intermittent positive-negative pressure respirator,* ventilating with 100 per cent oxygen and with a CO_2 absorption cannister in the circuit. The efficiency of dual phase ventilation was displayed in the low pCO_2 and elevated pH in many of the blood samples obtained prior to perfusion.[21] The resulting respiratory alkalosis appeared to have no adverse effects. During perfusion there was a fall in the buffer bicarbonate and a further drop in the pCO_2 (Table II). The effectiveness of CO_2 exchange by the oxygenator is indicated by the arteriovenous CO_2 difference.

TABLE II. CHANGES OF CO_2 AND pH BEFORE AND AT END OF PERFUSION

	BEFORE PERFUSION				END OF PERFUSION					
	ARTERIAL				ARTERIAL				VENOUS	
	CO_2 CONTENT (mM/L.)	pCO_2* (MM. HG)	HCO_3* (MEQ./L.)	pH	CO_2 CONTENT (mM/L.)	pCO_2* (MM. HG)	HCO_3* (MEQ./L.)	pH	CO_2 CONTENT (mM/L.)	A-V CO_2 DIFF. (mM/L.)
Average	13.74	32	15.1	7.32	8.52	18	9.5	7.36	14.50	5.98
S.D.	±5.86			±.65	±2.87			±.69	±3.58	±2.24
Range										
minimum	5.41	13	5.6	7.00	2.77	<10	6.2	7.00	8.15	3.06
maximum	25.41	86	26.1	7.73	18.77	35	13.9	7.73	25.62	14.44

*Determined from nomogram of Singer and Hastings.[31]

Comparison of the pyruvate levels in the donor reservoir and in the recipient animals prior to perfusion demonstrated an average increase of 209 per cent; the average rise during the by-pass was 21 per cent. This is shown graphically in Fig. 3. Blood chloride appeared to rise slightly† and was due, in part, to the incomplete removal of isotonic sodium chloride used for flushing the by-pass circuit free of formaldehyde. The values for free plasma hemoglobin exceeded the levels mentioned in previous reports from this laboratory.[7] The practice of replacing excessive operative losses with blood retrieved by suction (average free plasma hemoglobin—222 mg. per 100 ml.) was implicated. Total

*Air-Shields, Inc., Hatboro, Pa.
†P value for difference between control and perfusion samples was .06.

protein and fractions were not altered significantly by perfusion. This has been confirmed by plasmaphoretic studies.[22] Hematocrit and hemoglobin were unchanged (see Fig. 3).

In the presence of a normal, reduced, or augmented circulating volume in the recipient, the venous pressure was positive when the drainage capacity of the caval catheters was less than the return to the heart and was negative if the capacity of the catheters exceeded the return. Successful perfusions were conducted under both conditions. As the most sensitive indicator of variations in the circulating volume, the venous pressure responded to changes of less than 50 ml. Fluctuations in the flowmeter curve, level of blood in the oxygenator, blood pressure and stroke rate of the pumps corresponded, to a lesser degree, with those in the venous pressure. These indices were used to maintain the circulatory volume during perfusion.

The average blood loss was 1,050 ml. (range, 125 ml. to 2,300 ml.). The average replacement was 1,300 ml. Perfusion and postoperative chest losses averaged 630 and 370 ml., respectively. A comparison of pre- and postoperative weights revealed a positive blood balance in the majority of perfusions.

The clotting time in 22 animals was restored to normal in an average period of two hours by a single dose of protamine. Additional increments were required in 9 animals. Thirty of the 31 dogs had normal clotting times by the fifth postperfusion hour. The average initial dose of protamine required for neutralization of circulating heparin was 4.6 mg. per kilogram. The absence of stoichiometric neutralization of the original heparinizing dose was attributed to the variation in potency of protamine lots and emphasized the need for titrations to arrive at the correct dosage.

Table III summarizes the data on fibrillation. The reversibility of coronary air embolism is shown in Table IV and was in accord with the work of Geoghegan and Lam.[23] The results indicated that neither body positioning nor the introduction of a ventricular vent provided complete protection from this hazard. Coronary air embolism was not observed in any of the 6 dogs in which fibrillation was deliberately induced. Incision through the septum into the left atrium in this group was accompanied by a continuous, occasionally vigorous,

TABLE III. VENTRICULAR FIBRILLATION

CAUSE		PERIOD OF FIBRILLATION (MIN.)	DEFIBRILLATION	SURVIVOR	CAUSE OF DEATH
Placement of vent	1	28	1	1	
Coronary air embolism	4	14-41	3	2	Cerebral air embolism, cardiac standstill
Induced	6	14-34	6	6	

flow of arterialized blood from the left side. This was not observed in those hearts contracting regularly. Frequently, the blood level in the left atrium was displaced by air and remained below the incised septum. The diagnosis of coronary air embolism was established by direct inspection of coronary arteries and was confirmed by disturbances in rhythm, mottling and cyanosis

TABLE IV. AIR EMBOLISM

DOG NUMBER	SOURCE OF AIR	PROPHYLAXIS	ECG	RESULTS	SURVIVAL
83	Septal defect	Vent	Ventricular fibrillation	Coronaries cleared of air, defibrillated	Survivor
100	Septal defect	Vent	No change	Coronaries cleared of air	Died 12 hours postperfusion of hemorrhage
300*	Septal defect	Vent	Current of injury	Reverted to normal ECG	Died at operation of cerebral air embolism
201	Septal defect	Vent	No change	Coronaries cleared of air	Died 18 hours postperfusion of cerebral air embolism
433	Septal defect	Position	Ventricular fibrillation	Coronaries cleared of air, defibrillated	Died 48 hours postperfusion of cerebral air embolism
475	Septal defect	Position	Ventricular fibrillation	Coronaries cleared of air, defibrillated	Survivor
469	Catheter in left ventricle	Position	Ventricular fibrillation	Coronaries cleared of air, heart into standstill	Died at end of perfusion

*No air noted in coronary vessels.

of the myocardium. Clearance of air from the coronary vessels was accelerated by raising the perfusion pressure (increasing the air pressure activating the pumps), augmenting the circulating volume, cardiac massage, and compression of the aorta distal to the subclavian cannula. Three of the 6 animals whose hearts were returned to normal sinus rhythm succumbed to cerebral air embolism (see Table IV).

There were 22 survivors in thirty-one perfusions. Four of nine deaths were attributed to air embolism. One of the animals (433) survived for 48 hours and demonstrated opisthotonos, extensor rigidity, hyperreflexia, and hyperirritability. Vascular changes associated with cerebral air embolism were observed at autopsy.[24, 25] A dissecting aneurysm in one animal was ascribed to a tear in the intima of the left subclavian artery at the aortic junction, caused by a metal cannula. Two dogs died of intrapleural hemorrhage 12 hours postoperatively. Extensive damage to the interatrial septum and loss of an airway were responsible for two deaths.

There was a late mortality (more than 5 days postoperative) consisting of 10 dogs. These deaths, for the most part, were caused by wound infection with empyema. Two of these were secondary to sternal separations of transsternal incisions. One animal which was perfused through the femoral artery and vein developed congestive heart failure and pulmonary edema. Autopsy disclosed that a margin of the septal incision had been sutured to the "valve" of the inferior vena cava, shunting the blood from it into the left atrium. Severe stenosis of the inferior vena cava due to the atrial wall suture was the cause of death in another animal. The use of the right atrium for placement of caval catheters which serve as guides during suture, has prevented repetition of these errors. An infected thrombus adherent to the septal suture line was found in another animal with empyema.

Gross examination of the hearts of all but 2 of the 12 animals sacrificed at intervals up to eight months disclosed healed, smoothly endothelized intra-

cardiac incisions. A thickened mural plaque and a calcified pedunculated thrombus, both overlying the septal suture line, were seen in 2 animals. Inspection of the abdominal viscera revealed no evidence of recent or healed infarcts.

On the basis of these experimental investigations, the apparatus was employed in a clinical case.

CASE REPORT

J. B., an 18-year-old girl, a known "cardiac" since the age of one year, complained of limited exercise tolerance, exertional dyspnea, and precordial pain. On admission to the hospital, physical examination revealed a well-developed white female (53 Kg.) who presented a left precordial bulge. There was a harsh systolic murmur (Grade 3) heard loudest over the pulmonic area; the second pulmonic sound was diminished. No cyanosis or clubbing was noted. Chest x-ray showed enlargement of the right ventricle with a prominent left heart border. The electrocardiogram revealed a right ventricular strain pattern. Angiography demonstrated simultaneous opacification of the pulmonary artery and aorta. Catheterization data* are summarized in Table V. They were interpreted as

TABLE V. CATHETERIZATION DATA—PATIENT J. B.

	PREOPERATIVE		POSTOPERATIVE	
LOCATION OF SAMPLE	PRESSURE (MM. HG)	O_2 CONTENT (C.C./100 ML.)	PRESSURE (MM. HG)	O_2 CONTENT (C.C./100 ML.)
Pulmonary capillary	11	16.0	10	15.7
Pulmonary artery	20/12	15.4	23/13	11.9
Right ventricle (infundib.)	27/10	15.8	-	-
Right ventricle (sinus)	60/0	-	37/0	11.5
Right atrium	10	14.7	10	11.6
Superior cava	-	11.9	-	9.5
Brachial artery	-	16.0*	-	15.9†
Brachial artery (after exercise)	-	15.6‡		
Pulmonary blood flow	7.5 L./min.		5.8 L./min.	
Systemic blood flow	1.1 L./min.		5.1 L./min.	
Shunt, left-to-right	6.4 L./min.		0.7 L./min.	

*94.3 per cent saturated.
†94.5 per cent saturated.
‡92.0 per cent saturated.

indicating the presence of an interatrial septal defect with pulmonic stenosis of the infundibular type. The predominant shunt was left to right. On exercise, a slight right-to-left shunt was noted. On June 30, 1955, open heart surgery was performed through a transsternal incision. The patient underwent total body perfusion and an atrial cardiotomy was made in the excluded heart. A large foramen ovale type defect, 3 cm. by 5 cm., was obliterated with interrupted sutures. During the repair, a pool of blood was maintained over the defect to prevent air from entering the left side of the heart. Following closure of the right atrium, a 2 cm. incision was made in the right ventricular outflow tract and the right ventricle digitally explored. No infundibular stenosis was detected and the cardiotomy was closed. The patient's heart was excluded for 23 minutes during which time the extracorporeal flow rate averaged 36 ml. per kilogram per minute. The patient was awake at the conclusion of the procedure. Her postoperative course was uneventful and she was discharged on the thirtieth hospital day.

Six months later she was recatheterized. The data (see Table V) indicated a small left-to-right shunt (0.7 L. per minute) which was considered to be of no clinical significance. At this time she was free of exertional dyspnea and was engaging in unrestricted activity.

*Courtesy of Drs. Harold A. Lyons and John J. Kelly.

DISCUSSION

The results of these experiments show that the important metabolic changes occurring in a total by-pass are confined to the factors regulating acid-base balance. The appearance of a metabolic acidosis in perfused dogs has been attributed to a rise in fixed acid.[6, 26, 27] Pyruvic acid levels above 4 mg. per 100 ml. have been associated with a poor prognosis.[9] In this series, the increase in pyruvate was of minimal significance. The fall in bicarbonate buffer (see Table III) indicated that other acid metabolites were accumulating during perfusion. An appreciable increase in lactic acid has been reported.[26] Hyperventilation with a dual phase respirator in the preperfusion period and excessive elimination of CO_2 by the screen oxygenator during the by-pass produced a marked depression of the arterial pCO_2. This served to compensate for the metabolic acidosis, maintain the pH within normal limits and improve the oxygen uptake of hemoglobin (Bohr effect). Similar observations have been made with cross circulation techniques where an increase in the donor respiratory minute volume was used to control the fall in recipient pH.[27] Measures to regulate the pH by the addition of Na HCO_3 or flushing the oxygenator with 5 per cent CO_2 and 95 per cent oxygen were not employed.

Hemoglobinemia in hypotensive states has been recognized as a cause of renal damage in man.[28] However, under low perfusion pressures such as obtained in these experiments, no ill effects were noted even when the free plasma hemoglobin levels were in excess of 400 mg. per 100 ml. The recent adoption of a low-vacuum, variable suction pump has avoided foam formation and reduced the red cell destruction in the aspirated blood returned to the circuit.

In our experience the prime requisite for control of postperfusion hemorrhage and the restoration of normal clotting was complete reversal of heparinemia with protamine. Fractional protamine titrations obtained at regular intervals in the recovery period indicated reduced but significant concentrations of circulating heparin (5 to 10 mcg. per milliliter) which prevented normal clotting.[14] The existence of a heparin rebound phenomenon has been postulated to explain the persistence of circulating heparin despite adequate neutralizing doses of protamine.[4] The role of the fresh whole blood transfusion requires further evaluation. In early experiments it was given routinely at the completion of the by-pass to replace destroyed clotting factors, equalize postoperative losses and avoid the introduction of other anticoagulants. More recent evidence indicates that with a normal or slightly positive blood balance at the end of perfusion, adequate protamine dosage alone will restore the clotting time. Protracted perfusions (over one hour) appear to enhance postoperative bleeding. The hemorrhagic deaths in 2 dogs early in this series were ascribed to abnormal hemolytic and proteolytic activity due to plasma fibrinolysin[29] or bacterial contamination.[30] The impression has since been gained that these deaths can be averted by stricter attention to the details of postoperative care.

Exposure of the left side of the isolated contracting heart to the atmosphere directly or through septal defects predisposes to systemic air embolism. In 6 animals, incisions into the interatrial septum were accompanied by the entry of air into the left atrium and left ventricle in diastole. In the presence of a

competent mitral valve, bronchial artery return admixed with air was ejected into the systemic circulation in systole. Continued circulation through the left side of the heart made it difficult to preserve a level of blood or saline solution in the right atrium over the incised septum. Extracorporeal circuits which employ low perfusion pressures and deliver pulsatile flows are unable to maintain continuous closure of the aortic valves. Ventricular systolic pressure exceeds the perfusion pressure, especially in the diastolic phase of the mechanical pump, and ejection of air and blood into the aorta follows. Cookson and Costas-Durieux,[31] using hypothermia supplemented by arterial transfusions in open cardiotomies, stated that a nonpulsatile flow at a pressure of 70 mm. of Hg prevented opening of the aortic valves in ventricular systole; the technique was not uniformly effective in avoiding air embolism in their animals. Potassium-induced cardiac arrest has been advocated by Melrose and his group.[32] This method requires occlusion of the aorta above the coronary ostia, thereby limiting the period of by-pass and depriving the myocardium of the benefits of continuous perfusion. Miller and associates[15] have employed a left ventricular vent in experimental procedures and in a successful clinical case with an interatrial septal defect. In our hands, this method failed to eliminate coronary and/or cerebral air embolism in 4 of the 13 dogs in which it was used. Previous experience with 5 cases of ventricular fibrillation (4 due to coronary air embolism and one due to placement of a ventricular vent) had demonstrated the excellent protection afforded the heart by uninterrupted perfusion of the coronaries with oxygenated blood (see Table III). Accordingly, fibrillation to abolish ventricular ejection, as first proposed by Glenn and Sewell[33] and Senning,[10] was deliberately induced in 6 dogs with gratifying results. The "sucking action"[33] of the ventricle in diastole was replaced by a persistent flow of blood from the left atrium through the septal defect. Air embolism was not observed. Operative techniques were expedited by the quiet field. Myocardial tone and color remained satisfactory throughout the by-pass. There was a coarse fibrillatory pattern on the electrocardiogram. The hearts were defibrillated with ease soon after closure of the cardiac incisions. Promptness in restoring regular contractions reduced the tendency of the chambers to dilate because of the accumulation of coronary venous and bronchial artery blood. All 6 animals survived and at autopsy showed no myocardial damage attributable to fibrillation. The use of ventricular fibrillation as an adjunct to perfusion in the direct approach to lesions of the left side of the heart is currently being explored.

The available evidence indicates that the venous pressure is a valuable guide to the progress of a perfusion. This study will be presented in a separate paper.

SUMMARY

1. A simplified pump-oxygenator with safeguards against the introduction of air is described.

2. Interatrial septal defects were created and repaired under direct vision in 31 dogs with 22 survivors. During this study, techniques have been developed which appear to have reduced the hazards of total body perfusion.

3. Protamine titrations are an important factor in the control of post-perfusion bleeding.

4. The principal metabolic change during perfusion is the development of a metabolic acidosis. The desirability of a low arterial pCO_2 to counteract this is discussed.

5. The case report of a successful closure under direct vision of an interatrial septal defect in an 18-year-old girl is presented.

6. Deliberately induced ventricular fibrillation can eliminate air embolism during open cardiac surgery.*

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*Since the preparation of this report, induced fibrillation has been employed effectively in the direct vision repair of 2 clinical cases of interventricular septal defect.

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